PB82-205790

Effects of Continuous Chlorination on Spat of the American Oyster ('Crassostrea virginica')

(U.S.) Environmental Research Lab.
Gulf Breeze, FL

Dec 81

U.S. Department of Commerce
National Technical Information Service
NTIS

ERL,GB 049

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2.	3.
EPA-600/J-81-541	Journal Article	PB82-205790

4. TITLE AND SUBTITLE
EFFECTS OF CONTINUOUS, LOW-LEVEL CHLORINATION ON AMERICAN OYSTER (CRASSOSTREA VIRGINICA) SPAT

5. REPORT DATE
April 1982

6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)
Peter F. Sheridan

8. PERFORMING ORGANIZATION REPORT NO.

9. PERFORMING ORGANIZATION NAME AND ADDRESS

10. PROGRAM ELEMENT NO.

11. CONTRACT/GRANT NO.

12. SPONSORING AGENCY NAME AND ADDRESS
Environmental Research Laboratory
Office of Research and Development
U.S. Environmental Protection Agency
Gulf Breeze, Florida 32561

13. TYPE OF REPORT AND PERIOD COVERED

14. SPONSORING AGENCY CODE
EPA/600

15. SUPPLEMENTARY NOTES
Estuaries, Vol 4, No. 4, pp 376-378, December 1981

16. ABSTRACT

Newly settled spat of the American oyster (Crassostrea virginica) exhibited reduced survival and growth when exposed to chronic chlorination (as Na-OCl). Spat exposed to nominal concentrations of 0.250 and 0.500 mg per l chlorine-produced oxidant (CPO) had only 20% survival after 12 weeks, compared to 64% survival in controls. Spat growth was retarded by nominal concentrations as low as 0.125 mg per l CPO grew 39% and spat exposed to 0.250 mg per l CPO had no net growth. Surviving spat exposed to 0.500 mg per l CPO showed an apparent growth increase of 160%, due mainly to heavy mortality among samller-sized spat. The numbers of spat (whether surviving or not) that showed any growth over the 12-week exposure period decreased with increasing chlorine concentration. Larger spat were found to be more resistant than smaller spat to increased chlorination. The mechanism of growth inhibition in spat (mantle retraction and cessation of feeding) appeared to be similar to that found in adult oysters exposed to chlorine.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)*	21. NO. OF PAGES
RELEASE TO PUBLIC	UNCLASSIFIED	3
	20. SECURITY CLASS *(This page)* UNCLASSIFIED	22. PRICE

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA 22161

EPA-600/J-81-541
Journal Article

Effects of Continuous Chlorination on Spat of the American Oyster (*Crassostrea virginica*)[1]

ABSTRACT: Newly settled spat of the American oyster (*Crassostrea virginica*) exhibited reduced survival and growth when exposed to chronic chlorination (as NaOCl). Spat exposed to nominal concentrations of 0.250 and 0.500 mg per l chlorine-produced oxidant (CPO) had only 20% survival after 12 weeks, compared to 64% survival in controls. Spat growth was retarded by nominal concentrations as low as 0.125 mg per l CPO. Shell height of control oysters increased 103% after 12 weeks, while spat exposed to 0.125 mg per l CPO grew 39% and spat exposed to 0.250 mg per l CPO had no net growth. Surviving spat exposed to 0.500 mg per l CPO showed an apparent growth increase of 160%, due mainly to heavy mortality among smaller-sized spat. The numbers of spat (whether surviving or not) that showed any growth over the 12-week exposure period decreased with increasing chlorine concentration. Larger spat were found to be more resistant than smaller spat to increased chlorination. The mechanism of growth inhibition in spat (mantle retraction and cessation of feeding) appeared to be similar to that found in adult oysters exposed to chlorine.

Introduction

The American oyster, *Crassostrea virginica*, is a valuable economic and ecological resource (Wells 1961; Galtsoff 1964). Wastewater chlorination is an important public health process. However, discharge of chlorinated effluents into estuarine and coastal waters can cause toxic effects to many organisms, including oysters. This has spurred research concerning chlorination practices [see recent publications by Jolley (1978) and Jolley et al. (1978)]. Various investigations have addressed acute and chronic effects of chlorination on oysters, although the results are not truly intercomparable due to differing methodologies. Galtsoff (1946) found reduced pumping rates in adult oysters chlorinated at 0.05 mg per l free chlorine and valve closure in oysters exposed to 1.00 mg per l free chlorine or more for 48 hr. Bongers et al. (1977) found no mortality among oysters exposed for 15 days to 0.035 to 0.085 mg per l total residual oxidant (equivalent to total residual chlorine, TRC), although compared to controls, significantly less new shell deposition was observed. Scott and Middaugh (1978) examined the effects of chronic chlorination on adult oysters and demonstrated reduced survival at nominal 3.20 to 5.60 mg per l chlorine-produced oxidant (CPO; equivalent to TRC) as well as reduced fecal production and condition and gonadal indices at levels as low as nominal 1.0 mg per l CPO. Many of these effects were related to seasonal chlorine-temperature interactions (Scott and Vernberg 1979).

Less work has been reported on larval and juvenile (spat) stages. Waugh (1964) found no mortality of oyster (*Ostrea edulis*) larvae after intermittent chlorination at 5 mg per l TRC for 3 min. Roberts et al. (1975) documented 48-hr TL50 values for oyster larvae of 0.11 mg per l TRC in static tests and <0.005 mg per l TRC in constant addition tests. They also noted a 96-hr EC50 value of 0.023 mg per l TRC for spat, based on shell deposition. However, this EC50 value was estimated, not directly measured. The objective of the present study was to assess directly the effects of continuous chlorination on survival and growth of newly settled oyster spat during the summer, when chlorine-temperature interactions are known to produce the highest toxicity and greatest physiological stress in adult oysters (Scott et al. 1980).

Materials and Methods

Spat were collected on oyster shells suspended in We Creek, a tidal creek adjacent to the Bears Bluff Field Station on Wadmalaw Island, 40 km south of Charleston, South Carolina (lat. 32°40′N, long. 80°18′W). After 6 weeks (16 May to 28 June 1979), the shells were removed from the creek, cleaned of all encrusting organisms except spat, and suspended in an outdoor, flow-through microcosm system described in detail by Davis et al. (1977). The containers (38-liter capacity) received 40 l per hr (1.6 l per hr per spat) unfiltered estuarine water pumped continuously from the adjacent North Edisto River. Temperature and salinity in the system were monitored daily during the exposure period; temperatures ranged from 21 to 33 °C ($\bar{x}$ = 27.8 °C), salinities ranged from 22 to 35‰ ($\bar{x}$ = 27.7‰).

Twenty-five oyster spat were suspended in each of four containers that received analytical grade NaOCl continuously applied by syringe pumps at nominal concentrations of 0.000, 0.125, 0.250, and 0.500 mg per l CPO mixed at the delivery head. Reported chlorine concentrations employ additions of known amounts of aqueous solutions of chlorine, whereas measurement of "chlorine residuals" by amperometric titration actually detects activities of a wide variety of chlorine-produced oxidants without identification or quantification of oxidant species. Chlorine residuals were not measured during this study, since these chlorine concentrations typically produce no detectable (by amperometric titrator) chlorine residuals during summer months (Davis et al. 1977). Extrapolation of potential residual concentrations from measurements made on nominal chlorine concentrations of 1.0 to 5.6 mg per l CPO during previous summer studies (D. P. Middaugh, U.S. Environmental Protection Agency, Bears Bluff Field Station, personal communication) indicated the following: 0.125 mg per l CPO produced 0.003 mg per l residuals, 0.250 mg per l CPO produced 0.01 mg per l residuals, and 0.500 mg per l CPO produced 0.02 mg per l CPO residuals. Spat were exposed for 12 weeks (28 June to 20 September 1979). Spat survival

[1] Contribution No. 411, U.S. Environmental Protection Agency, Environmental Research Laboratory Gulf Breeze, Florida 32561.

0160-8347/81/040376-03$01.50/0

TABLE 1. Survival and growth of oyster (*Crassostrea virginica*) spat after 12 weeks of continuous chlorination (as NaOCl). N = 25 spat per concentration at week 0.

Response	Nominal CPO (mg per l)	Week 0	2	4	6	8	12
Numbers surviving	0.000	25	25	24	22	18	16
	0.125	25	25	23	23	21	17
	0.250	25	24	24	16	10	5[a]
	0.500	25	25	19	14	12	5[a]
Mean shell height (and range), mm	0.000	5.2 (2–8)	5.8 (2–8)	6.1 (2–8)	7.1 (4–12)	8.3 (5–13)	10.6 (6–15)
	0.125	6.6 (4–11)	6.8 (4–12)	7.3 (4–13)	7.4 (4–13)	7.5 (4–13)	9.1 (6–15)
	0.250	6.2 (3–9)	6.4 (3–9)	6.5 (3–10)	6.7 (3–10)	7.0 (4–10)	6.2 (4–9)
	0.500	4.8 (2–8)	5.0 (3–8)	5.6 (4–9)	5.6 (4–9)	6.2 (4–11)	12.6 (9–15)
Cumulative percentage increase in mean shell height from week 0	0.000	—	10.7	17.0	36.3	58.0	102.7
	0.125	—	3.7	10.7	12.0	14.6	39.0
	0.250	—	3.5	6.2	8.6	13.6	0.7
	0.500	—	3.3	16.3	15.1	27.5	160.3

[a] Significantly different from controls (χ^2, $p < 0.01$).

and growth (as shell height) were recorded at two-week intervals beginning at week 0 (except week 10). During sampling periods, the shells were cleaned of encrusting organisms and the containers drained and flushed to prevent build-up of sediments, algae, and potential predators.

Results and Discussion

Oyster spat were found to have several responses to the various chlorination regimes over the 12-week period (Table 1). Survival of spat exposed to nominal 0.125 mg per l CPO was similar to controls, but survival of spat exposed to nominal 0.250 and 0.500 mg per l CPO decreased significantly (χ^2, $p < .01$) after 12 weeks. Although decreased survival was not statistically significant until week 12, it was apparent by week 6 among spat that received 0.250 mg per l CPO and by week 4 in spat that received 0.500 mg per l CPO (Table 1).

Spat growth was also affected by continuous chlorination. Mean shell height in control oysters doubled during the exposure period. Shell growth in spat exposed to 0.125 mg per l CPO was retarded and spat exhibited only a 39% increase after 12 weeks (controls had shown a similar increase by week 6). Spat receiving 0.250 mg per l CPO showed no net increase in shell height after 12 weeks. By week 6, when survival was similar to controls at week 12, these spat had grown less than the control spat had by week 2. Shell height of spat exposed to 0.500 mg per l CPO increased 160% by week 12. This apparent anomaly was caused by reduced survival among smaller-sized spat such that the final size range was skewed towards the larger size classes (9–15 mm in 0.500 mg per l CPO, compared with 6–15 mm in controls at week 12, both beginning at week 0 with ranges of 2–8 mm).

These results indicated both lethal and sublethal effects of continuous chlorination upon oyster spat. At a chlorine concentration of 0.125 mg per l CPO, growth was retarded but survival was not affected. An exposure concentration of 0.250 mg per l CPO inhibited growth even more and also decreased survival of all sizes of spat tested, whereas at 0.500 mg per l CPO, the smaller spat were eliminated. In fact, the survivors of 0.500 mg per l CPO were 33% larger at week 0 than spat that died over the 12-week period (Table 2). Such was not the case for other concentrations, in which spat that survived to week 12 were the same size (−3 to +8% size differential) as non-survivors at week 0. Larger spat were more resistant than smaller spat to increased chlorination. Also, the proportion of all spat (survivors and non-survivors) that showed any

TABLE 2. Growth responses of oyster spat exposed to 12 weeks of continuous chlorination. N = 25 spat per concentration at week 0.

Response	Nominal CPO (mg per l) 0.000	0.125	0.250	0.500
Mean size (mm) of week 12 survivors at week 0 (A)	5.3	6.9	6.0	6.0
Mean size (mm) of nonsurvivors at week 0 (B)	5.0	6.3	6.2	4.5
Percentage difference (A/B)	+6.2	+8.5	−3.0	+33.0
Percentage of all spat showing growth	80	60	36	44

growth decreased as chlorine concentration increased (80% in controls, 60% in 0.125 mg/liter CPO, 36% in 0.250 mg per l CPO, and 44% in 0.500 mg per l CPO).

This experiment indicates that continuous chlorination of estuarine waters at a nominal concentration of 0.125 mg per l CPO retards growth of newly settled oyster spat and that nominal concentrations of 0.250 to 0.500 mg per l CPO become lethal. By way of comparison, current practices at coastal power generating stations employ continuous or intermittent chlorination levels of 1 to 2 mg per l CPO (Coughlan and Whitehouse 1977; White 1978). Chronic exposure to these concentrations does not impair survival of adult oysters, but does decrease physiological condition, accumulation of gonadal material, and feeding activity, as indicated by fecal production (Scott and Middaugh 1978). Fecal production in adult oysters also decreases in response to nominal chlorine concentrations as low as 0.18 mg per l CPO (G. I. Scott, U.S. Environmental Protection Agency, Bears Bluff Field Station, personal communication). Recent studies by Scott et al. (1980) report that exposure of adult oysters to chlorination causes the mantle to retract from the valve edge and to deposit a new growth layer in the pallial line region, thereby inhibiting valve growth. Preliminary examination of spat exposed to chlorination in the present study indicated that a mantle retraction layer was present. These findings suggest that typical chlorination rates or process by-products or both are lethal to oyster spat either directly or indirectly by prevention of feeding and growth.

Literature Cited

Bongers, L. H., B. Bradley, D. T. Burton, A. F. Holland, and L. H. Liden. 1977. Biotoxicity of bromine chloride and chlorine treated power plant condenser cooling water effluents. Martin Marietta Corp., Environ. Tech. Center, Baltimore, Md. 61 p.

Coughlan, J., and J. Whitehouse. 1977. Aspects of chlorine utilization in the United Kingdom. *Chesapeake Sci.* 18:102–111.

Davis, W. P., B. S. Hester, R. L. Yoakum, and R. G. Domey. 1977. Marine ecosystem testing units: design for assessment of benthic organism responses to low level pollutants. *Helgol. Wiss. Meeresunters.* 30:673–681.

Galtsoff, P. S. 1946. Reactions of oysters to chlorination. U.S. Dept. Interior, Fish Wild. Serv., Research Rept. 11. 28 p.

Galtsoff, P. S. 1964. The American oyster *Crassostrea virginica* Gmelin. *U.S. Fish. Wildl. Serv., Fish. Bull.* 64:1–480.

Jolley, R. L. 1978. Water Chlorination: Environmental Impact and Health Effects, Vol. 1. Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan. 439 p.

Jolley, R. L., H. Gorchev, and D. H. Hamilton, Jr. 1978. Water Chlorination: Environmental Impact and Health Effects, Vol. 2. Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan. 909 p.

Roberts, M. H., Jr., R. J. Diaz, M. E. Bender, and R. J. Huggett. 1975. Acute toxicity of chlorine to selected estuarine species. *J. Fish. Res. Board Can.* 32:2525–2528.

Scott, G. I., and D. P. Middaugh. 1978. Seasonal chronic toxicity of chlorination to the American oyster, *Crassostrea virginica* (G), p. 311–328. *In* R. L. Jolley, H. Gorchev, and D. H. Hamilton, Jr. (eds.), Water Chlorination: Environmental Impact and Health Effects, Vol. 2. Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan.

Scott, G. I., D. P. Middaugh, N. Watabe, A. M. Crane, and N. H. McGlothlin. 1980. Physiological effects of chlorine-produced oxidants and uptake of chlorination by-products in the American oyster, *Crassostrea virginica* (Gmelin), p. 501–516. *In* R. L. Jolley, W. A. Brungs, and R. B. Cumming (eds.), Water Chlorination: Environmental Impact and Health Effects, Vol. 3. Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan.

Scott, G. I., and W. B. Vernberg. 1979. Seasonal effects of chlorine-produced oxidants on the growth, survival, and physiology of the American oyster, *Crassostrea virginica* (Gmelin), p. 415–435. *In* W. B. Vernberg, F. P. Thurberg, A. Calabrese, and F. J. Vernberg (eds.), Marine Pollution: Functional Responses. Academic Press, Inc., New York.

Waugh, G. D. 1964. Observations on the effects of chlorine on the larvae of oysters, *Ostrea edulis* L., and barnacles, *Elminius modestus* Darwin. *Ann. Appl. Biol.* 54:423–440.

Wells, H. W. 1961. The fauna of oyster beds, with special reference to the salinity factor. *Ecol. Monogr.* 31:239–266.

White, G. C. 1978. Current chlorination and dechlorination practices in the treatment of potable water, wastewater and cooling water, p. 1–18. *In* R. L. Jolley (ed.), Water Chlorination: Environmental Impact and Health Effects, Vol. 1. Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan.

Peter F. Sheridan[2]
U.S. Environmental Protection Agency
Gulf Breeze Environmental Research Laboratory
Bears Bluff Field Station
Box 368
Johns Island, South Carolina 29455

[2] Present address: National Marine Fisheries Service, Southeast Fisheries Center, 4700 Avenue U., Galveston, Texas 77550.